```
FILE 'MEDLINE'
FILE 'JAPIO'

FILE 'BIOSIS'

FILE 'SCISEARCH'

FILE 'WPIDS'

FILE 'CAPLUS')

FILE 'EMBASE'
=> s cd81
L1          1886 CD81

=> s l1 and degranulation
L2            20 L1 AND DEGRANULATION

=> s l2 and inflammatory or inflamation
L3           442 L2 AND INFLAMMATORY OR INFLAMATION

=> s l2 and (inflammatory or inflamation)
L4             4 L2 AND (INFLAMMATORY OR INFLAMATION)

=> s l3 and allergic
L5            22 L3 AND ALLERGIC

=> s l4 and l5
L6             1 L4 AND L5

=> s l1 and (anti-cd81 antibody or cd81 antibody)
L7            53 L1 AND (ANTI-CD81 ANTIBODY OR CD81 ANTIBODY)

=> s l7 and degranulation
L8             6 L7 AND DEGRANULATION

=> s l8 and(astma or hay fever or atopic eczema)
L9             1 L8 AND(ASTMA OR HAY FEVER OR ATOPIC ECZEMA)

=> s l1 and(astma or hay fever or atopic eczema)
L10            2 L1 AND(ASTMA OR HAY FEVER OR ATOPIC ECZEMA)

=> s l1 and (passive cutaneous anaphylaxis)
L11            6 L1 AND (PASSIVE CUTANEOUS ANAPHYLAXIS)

=> dup rem l4
PROCESSING COMPLETED FOR L4
L12            2 DUP REM L4 (2 DUPLICATES REMOVED)

=> dup rem l5
PROCESSING COMPLETED FOR L5
L13           20 DUP REM L5 (2 DUPLICATES REMOVED)

=> dup rem l7
PROCESSING COMPLETED FOR L7
L14           18 DUP REM L7 (35 DUPLICATES REMOVED)

=> dup rem l8
PROCESSING COMPLETED FOR L8
L15            2 DUP REM L8 (4 DUPLICATES REMOVED)

=> dup rem l10
PROCESSING COMPLETED FOR L10
L16            1 DUP REM L10 (1 DUPLICATE REMOVED)

=> dup rem l11
PROCESSING COMPLETED FOR L11
L17            2 DUP REM L11 (4 DUPLICATES REMOVED)
```

=> d ibib abs l9

L9 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1998:402338 CAPLUS
DOCUMENT NUMBER: 129:66839
TITLE: Calcium-independent modulation by ***CD81*** of receptor signalling
INVENTOR(S): Fleming, Tony; Kinet, Jean-Pierre
PATENT ASSIGNEE(S): Beth Israel Deaconess Medical Center, USA
SOURCE: PCT Int. Appl., 63 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
WO 9825647	A1	19980618	WO 1997-US22743		19971209
W: AU, CA, JP					
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE					
US 2002004210	A1	20020110	US 1997-954279		19971020
US 6423501	B2	20020723			
AU 9855220	A1	19980703	AU 1998-55220		19971209
EP 948354	A1	19991013	EP 1997-951630		19971209
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI					
US 2002182726	A1	20021205	US 2001-4562		20011205
PRIORITY APPLN. INFO.:			US 1996-32963P	P	19961213
			US 1997-954279	A	19971020
			WO 1997-US22743	W	19971209

AB Calcium-independent ***CD81*** inhibition of IgE-mediated ***degranulation*** in mast cells, particularly through the Fc.gamma.RIII and Fc.epsilon.RI receptors, is described, as well as methods of inhibiting allergic processes. The method uses monoclonal ***anti*** - ***CD81*** ***antibody*** .
REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS . RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> d ibib abs l12 1-2

L12 ANSWER 1 OF 2 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 2003594622 MEDLINE
DOCUMENT NUMBER: PubMed ID: 14675193
TITLE: Generation of a large number of connective tissue type mast cells by culture of murine fetal skin cells.
AUTHOR: Yamada Nobuo; Matsushima Hironori; Tagaya Yutaka; Shimada Shinji; Katz Stephen I
CORPORATE SOURCE: Dermatology Branch, National Cancer Institute, National Institutes of Health, Bethesda, Maryland 20892, USA.
SOURCE: Journal of investigative dermatology, (2003 Dec) 121 (6) 1425-32.
Journal code: 0426720. ISSN: 0022-202X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200401
ENTRY DATE: Entered STN: 20031217
Last Updated on STN: 20040130
Entered Medline: 20040129
AB We describe a novel culture system for generating large numbers of murine skin-associated mast cells and distinguish their characteristics from bone marrow-derived cultured mast cells. Culture of day 16 fetal skin single cell suspensions in the presence of interleukin-3 and stem cell factor allowed expansion and maturation of mast cells in the presence of stromal cells. The average yield of mast cells after 2 wk was 7.3 million cells per fetus at a purity of 96%. These fetal skin-derived cultured mast cells increased their histamine content in a time-dependent manner to 3.6 pg per cell after 2 wk and 6.7 pg per cell after 4 wk. Phenotypic

analyses revealed much greater expression of CD49b and ***CD81*** and lesser expression of CD77 and CD102 on fetal skin-derived cultured mast cells as compared with bone marrow-derived cultured mast cells. These findings suggest a close similarity between fetal skin-derived cultured mast cells and freshly isolated cutaneous mast cells. Connective tissue mast cell characteristics of fetal skin-derived cultured mast cells were evidenced by: (1) their greater histamine content than bone marrow-derived cultured mast cells; (2) the presence of heparin; and (3) their ***degranulation*** in response to compound 48/80 and substance P. Importantly, fetal skin-derived cultured mast cells secreted greater amounts of interleukin-13 but much less MIP-1beta and interleukin-6 than bone marrow-derived cultured mast cells in response to ionomycin. Thus fetal skin-derived cultured mast cells have many characteristics distinct from bone marrow-derived cultured mast cells and can be used as a model of cutaneous mast cells to discern their functions.

L12 ANSWER 2 OF 2 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN
ACCESSION NUMBER: 1998-348267 [30] WPIDS
DOC. NO. NON-CPI: N1998-271821
DOC. NO. CPI: C1998-107646
TITLE: Modulation of ***CD81*** -mediated signal transduction - used for the treatment of e.g. allergic conditions, anaphylactic reactions, autoimmune disorders or bacterial or parasite infections.
DERWENT CLASS: B04 D16 S03
INVENTOR(S): FLEMING, T; KINET, J
PATENT ASSIGNEE(S): (BETH-N) BETH ISRAEL DEACONESS MEDICAL CENT; (FLEM-I) FLEMING T; (KINE-I) KINET J
COUNTRY COUNT: 22
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 9825647	A1	19980618	(199830)*	EN	62
RW: AT BE CH DE DK ES FI FR GB GR IE IT LU MC NL PT SE					
W: AU CA JP					
AU 9855220	A	19980703	(199847)		
EP 948354	A1	19991013	(199947)	EN	
R: AT BE CH DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE					
US 2002004210	A1	20020110	(200208)		
US 6423501	B2	20020723	(200254)		
US 2002182726	A1	20021205	(200301)		

APPLICATION DETAILS:

PATENT NO	KIND		APPLICATION	DATE
WO 9825647	A1		WO 1997-US22743	19971209
AU 9855220	A		AU 1998-55220	19971209
EP 948354	A1		EP 1997-951630	19971209
			WO 1997-US22743	19971209
US 2002004210	A1	Provisional	US 1996-32963P	19961213
			US 1997-954279	19971020
US 6423501	B2	Provisional	US 1996-32963P	19961213
			US 1997-954279	19971020
US 2002182726	A1	Provisional	US 1996-32963P	19961213
		Cont of	US 1997-954279	19971020
			US 2001-4562	20011205

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 9855220	A	Based on	WO 9825647
EP 948354	A1	Based on	WO 9825647

PRIORITY APPLN. INFO: US 1997-954279 19971020; US 1996-32963P 19961213; US 2001-4562 20011205
AN 1998-348267 [30] WPIDS
AB WO 9825647 A UPAB: 19991122

A calcium independent (CI) method of inhibiting cell surface receptor (CSR)-mediated signalling and/or ***degranulation*** (in a mammal), comprises contacting a cell (of the mammal) with an agent which induces ***CD81*** -mediated signal transduction (ST).

Inhibitors of ***CD81*** -mediated ST can be used conversely to enhance CSR-mediated signalling and/or ***degranulation*** .

USE - The methods can be used for the treatment of allergic conditions, e.g. asthma, hay fever or atopic eczema, anaphylactic reactions and related diseases. They can be used to treat allergic or ***inflammatory*** responses associated with disorders such as autoimmune diabetes mellitus, rheumatoid arthritis, ankylosing spondylitis, sarcoidosis, Sjogren's syndrome, multiple sclerosis, ***inflammatory*** bowel disease (i.e. Crohn's disease and ulcerative colitis), dermatomyositis, scleroderma, polymyositis, systemic lupus erythematosus, biliary cirrhosis, autoimmune thyroiditis, and autoimmune hepatitis, as well as many dermatological disorders, including psoriasis, contact sensitivity and atopic dermatitis. Enhancement of the cell surface receptors which induce mast cell ***degranulation*** is useful in inducing an ***inflammatory*** response, e.g. in response to bacterial or parasite infection. They can also be used to study receptor-mediated signalling in cells and to improve the therapeutic capability to modulate the function of such cells.

Dwg.0/12

=> d ibib abs l13 1-20

L13 ANSWER 1 OF 20 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED. on STN

ACCESSION NUMBER: 2004521930 EMBASE

TITLE: [Functional gastrointestinal disorders in food alergy in infants and young children].
ZABURZENIA CZYNNOSCIOWE PRZEWODU POKARMOWEGO W ALERGII POKARMOWEJ U NIEMOWLAT I MAlYCH DZIECI.

AUTHOR: Wasowska-Krolikowska K.; Ploceck A.; Toporowska-Kowalska E.

CORPORATE SOURCE: Dr. K. Wasowska-Krolikowska, Klin. Gastroeterol./Alergol. Dziec., Instytutu Pediatrii, Uniw. Szpital Kliniczny Nr 4 UM, ul. Sporna 36/50, 91-738 Lodz, Poland.
etka@csk.am.lodz.pl

SOURCE: Pediatria Wspolczesna, (2004) 6/4 (435-438).
Refs: 26
ISSN: 1507-5532 CODEN: PWESBM

COUNTRY: Poland

DOCUMENT TYPE: Journal; General Review

FILE SEGMENT: 007 Pediatrics and Pediatric Surgery
026 Immunology, Serology and Transplantation
048 Gastroenterology

LANGUAGE: Polish

SUMMARY LANGUAGE: Polish; English

AB Functional gastrointestinal disorders (FGID) in children ecomprise chronic or recurrent gastrointestinal abnormaliteis characterised by specific clinical manifestation which can not be explained by anatomic or biochemic abnormalities. Etiology of FGID remains unclear, food allergy beeing one of the possible causative agents. Immunologically mediated adverse reactions to foods can evoke chronic ***inflamation*** of the gut mucosa and, as the consequence, numerous defined clinical syndromes (immediate food hypersensitivity, alergic gastritis/enteritis/colitis, enteropathy, food allergy syndrome, eosinophilic gut inflammation) and clinical conditions with possible ***allergic*** mechanism (gastroesophageal reflux GER, irritable bowel syndrome IBS, infant colic, chronic constipation) as well.

L13 ANSWER 2 OF 20 CAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 2004:496185 CAPLUS

DOCUMENT NUMBER: 141:98858

TITLE: Protective role of activated protein C in lung and airway remodeling

AUTHOR(S): Suzuki, Koji; Gabazza, Esteban Cesar; Hayashi, Tatsuya; Kamada, Haruhiko; Adachi, Yukihiko; Taguchi, Osamu

CORPORATE SOURCE: Department of Molecular Pathobiology, Mie University

School of Medicine, Tsu-city, Mie, Japan
SOURCE: Critical Care Medicine (2004), 32(5, Suppl.), S262-S265
CODEN: CCMDC7; ISSN: 0090-3493
PUBLISHER: Lippincott Williams & Wilkins
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

AB A review. Recent studies have implicated the protein C pathway in the mechanism of lung and airway remodeling. The effector enzyme of this pathway is activated protein C (APC). Clin. studies have shown that APC generation is decreased in patients with lung injury and airway inflammation and that this decrease is assocd. with increased collagen deposition in the lung. In line with these findings, low APC activity has been obsd. in the bronchoalveolar lavage fluid in animal models of lung injury and airway inflammation. Treatment with APC significantly inhibits the development of lung fibrosis in bleomycin-induced lung injury and the development of airway hyperresponsiveness and ***allergic*** inflammation in ovalbumin-induced bronchial asthma. APC may protect the lung from fibrosis and airway remodeling by suppressing activation of coagulation, decreasing the secretion of inflammatory cytokines and platelet-derived growth factor, and promoting fibrinolysis. APC inhibits the expression of cytokines by decreasing the nuclear translocation of signal transducer and activator of transcription 6 and the nuclear factor-.vkappa.B family of transcription factors. In view of its multiple functions, APC constitutes a potential therapeutic agent for inflammatory disorders of the lung and airways.

REFERENCE COUNT: 28 THERE ARE 28 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 3 OF 20 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2003:173370 CAPLUS
DOCUMENT NUMBER: 138:210328
TITLE: Anti-inflammatory oxytocin formulations
INVENTOR(S): Uvnaes-Moberg, Kerstin; Lundeberg, Thomas
PATENT ASSIGNEE(S): Swed.
SOURCE: PCT Int. Appl., 65 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003017922	A2	20030306	WO 2002-SE1560	20020902
WO 2003017922	A3	20031009		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
EP 1432434	A2	20040630	EP 2002-763166	20020902
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK				

PRIORITY APPLN. INFO.: SE 2001-2910 A 20010831
WO 2002-SE1560 W 20020902

OTHER SOURCE(S): MARPAT 138:210328

AB The present invention relates to the use of substances with oxytocin for the prepn. of pharmaceutical compn. against ***inflamation*** . It also relates to a pharmaceutical compn. comprising at least one substance with oxytocin activity against ***inflamation*** .

L13 ANSWER 4 OF 20 MEDLINE on STN
ACCESSION NUMBER: 2003462936 MEDLINE
DOCUMENT NUMBER: PubMed ID: 14524284
TITLE: [Cytokines and anti-cytokines in ***allergic***

diseases].
Cytokiny i antycytokiny w chorobach alergicznych.
AUTHOR: Fal Andrzej M
CORPORATE SOURCE: Katedra i Klinika Chorob Wewnetrznych i Alergologii Akademii Medycznej we Wroclawiu.
SOURCE: Polski merkuriusz lekarski : organ Polskiego Towarzystwa Lekarskiego, (2003 Jun) 14 (84) 613-6. Ref: 37
Journal code: 9705469. ISSN: 1426-9686.
PUB. COUNTRY: Poland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, ACADEMIC)
LANGUAGE: Polish
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200401
ENTRY DATE: Entered STN: 20031004
Last Updated on STN: 20040115
Entered Medline: 20040114

AB ***Allergic*** ***inflamation*** is complexed phenomenon related to the activity of many mediators released from "effector cells". The role of IL-12, IL-5, IL-4 and some adhesive molecules is presented with special attention focused on therapeutical aspects in ***allergic*** diseases.

L13 ANSWER 5 OF 20 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on STN
ACCESSION NUMBER: 2003:156626 BIOSIS
DOCUMENT NUMBER: PREV200300156626
TITLE: Registered occupational diseases among personnel of Polish hospitals, 2001.
Original Title: Choroby zawodowe pracownikow szpitali, 2001..
AUTHOR(S): Peplonska, Beata [Reprint Author]; Szeszenia-Dabrowska, Neonila [Reprint Author]
CORPORATE SOURCE: Sw. Teresy 8, 90-950, Lodz, Poland
beatap@imp.lodz.pl
SOURCE: Medycyna Pracy, (2002) Vol. 53, No. 5, pp. 369-374. print.
CODEN: MEPAAX. ISSN: 0465-5893.
DOCUMENT TYPE: Article
LANGUAGE: Polish
ENTRY DATE: Entered STN: 26 Mar 2003
Last Updated on STN: 26 Mar 2003

AB The paper presents the data provided by the Central Register of Occupational Diseases in Poland on the compensated occupational diseases among hospital personnel, registered in 2001. The trends in the incidence of occupational diseases in this population over the period 1994-2001 are also discussed. In total, 394 new cases of occupational diseases among the hospital personnel were registered in 2001, which makes up 52.1% of all cases recorded under the "Health and social work" section of occupational activities, according to the Nomenclature des Activitees de Communite Europeene. Most of these cases were found among nurses (47%), followed by physicians (15%), laboratory analysts (11.5%), orderlies (11%), and dentists (3%) and referred mainly to females (84.8%). Contagious and invasive diseases prevailed, constituting 73.9% of all cases. Viral hepatitis made up 72.5% of all registered contagious and invasive diseases: HBV was diagnosed in 46%, HCV in 50.2% and HBV+HCV in 1.8% of all viral hepatitis cases. Dermatoses, mostly of ***allergic*** etiology, were the second most prevalent diseases (11.4%), and were most frequently associated with exposure to latex, thiurams, mercaptobenzothiazole and non-specified rubber compounds - 73% of all factors causing ***allergic*** dermatoses. Chronic diseases of locomotor system, chronic diseases of peripheral nervous system, chronic diseases of bronchi, chronic ***inflamation*** of nose, pharynx, larynx and trachea, and intoxications were also reported. Almost twofold decrease in the incidence rate in the population of workers referred to "Health and social work" activity section was observed in 2001 compared to 1994. The decrease in the number of the registered: occupational diseases seen in the hospital employees was mostly due to the effective anti HBV prevention programs carried out in Poland among health care personnel since 1989.

L13 ANSWER 6 OF 20 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
ACCESSION NUMBER: 2003042354 EMBASE
TITLE: [Procalcitonin].
PROKALSITONIN.
AUTHOR: Onal Z.; Onal H.; Yildiz E.; Yildiz C.K.; Siraneci R.
CORPORATE SOURCE: Dr. Z. Onal, SSK Bakirkoy Obstetric Train. Hosp., Department of Pediatrics, Istanbul, Turkey
SOURCE: SENDROM, (1 Dec 2002) 14/12 (81-90).
Refs: 72
ISSN: 1016-5134 CODEN: SENDEY
COUNTRY: Turkey
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 003 Endocrinology
004 Microbiology
LANGUAGE: Turkish
SUMMARY LANGUAGE: English
AB Recently a new marker called procalcitonin has added to the list of infectious disease markers. As a calcitonin propeptide procalcitonin is produced by C cells in tiroid gland. PCT can be used as a marker of systemic enflematory states triggered by bacterial infections (sepsis, septic shock). Infections that is not systemic or localized to an organ give rise to mild elevations in PCT level. Bacterial toxins play the major role in PCT production. Disease states that has bacterial toxins inits etiology (sepsis, septic schock, multiple organ disfunction) is characterized by such an high PCT levels like 10 to 100 ng/ml. Increase in PCT levels during disease states otoimmun neoplastic and viral infections ones are mild. Chronic non bacterial ***inflamations*** and ***allergic*** reactions have uncharged PCT level. These important properties of PCT is not possessed by many other infection markers.

L13 ANSWER 7 OF 20 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on STN
ACCESSION NUMBER: 2002:565684 BIOSIS
DOCUMENT NUMBER: PREV200200565684
TITLE: Efficacy of xidiphone in the combined treatment of atopic bronchial asthma in children.
AUTHOR(S): Pogomiy, N. N.; Svyatkina, O. B.; Pankova, G. F.; Chernova, O. I.
SOURCE: Rossiiskii Vestnik Perinatologii i Pediatrii, (2002) Vol. 47, No. 1, pp. 33-37. print.
DOCUMENT TYPE: Article
LANGUAGE: Russian
ENTRY DATE: Entered STN: 7 Nov 2002
Last Updated on STN: 30 Dec 2002
AB The paper outlines a new method of basic therapy for atopic bronchial asthma in infants and children - the oral use of xidiphone in doses of 18-42 mg per kg body weight during 4-6 weeks. A total of 507 children, including 132 infants aged 7 months to 3 years and 375 children aged 4 to 14 years, who had severe (n=89) and moderate (n=418) atopic bronchial asthma that was concurrent with pollinosis in 123 children. Xidiphone monotherapy was performed in 294 (58%) children, 213 children were on combined therapy (xidiphone and euphylline preparations), 29 children received xidiphone+corticosteroid hormones, 23 children on xidiphone received specific immunotherapy. Analysis of the efficacy of the drug revealed positive clinical changes in 315 children aged 4-14 years and in 123 infants: there was diminished bronchial obstruction, less frequency and disappearance of hard breathing attacks, prevention of seasonal manifestations of the disease; hormone therapy could be discontinued in infants taking prednisolone. Four-week xidiphone monotherapy caused increases in forced expiratory volume and forced inspiratory and expiratory volumetric velocities in children. In addition to positive changes in the clinical course of the disease, normalized membranous fluidity and suppressed transmembranous transposition of calcium ions in the lymphocytes, inhibited basophilic degranulation and leukocytic release of leukotrienes upon in vitro exposure to a specific allergen, substantially increased population of peripheral T-lymphocytic suppressors, and lower serum levels of total immunoglobulins E were observed. Thus, xidiphone treatment leads to correction of the functions of different cells, by preventing escalation of ***allergic*** ***inflamation*** , by promoting disappearance or alleviation of clinical

signs of the disease. The proposed treatment of atopic bronchial asthma is protected by the authors' certificate (No. 1680186 of June 1, 1991) as an original therapeutical finding.

L13 ANSWER 8 OF 20 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
ACCESSION NUMBER: 2002189189 EMBASE
TITLE: [Etiopathogeny of otitis media with effusion in a pediatric population].
ETIOPATOGENIA DA OTITE MEDIA COM EFUSAO NUMA POPULACAO PEDIATRICA.
AUTHOR: Lopes I.; Aleves E.; Soares T.; Coutinho M.; Teixeira F.
CORPORATE SOURCE: I. Lopes, Servico de Imunoalergologia, Hospital Maria Pia, Rua da Boavista, 827, 4050-111 Porto, Portugal
SOURCE: Nascer e Crescer, (2002) 11/1 (8-12).
Refs: 22
ISSN: 0872-0754 CODEN: NACRF7
COUNTRY: Portugal
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 007 Pediatrics and Pediatric Surgery
011 Otorhinolaryngology
026 Immunology, Serology and Transplantation
LANGUAGE: Portuguese
SUMMARY LANGUAGE: English; Portuguese
AB Introduction - Otitis media with effusion (OME) is a very common disease in children, and a frequent cause of hearing loss. Etiopathogenesis is still controversial, perhaps multifactorial, involving eustachian tube dysfunction, infection and nasal ***inflamation*** . Aim - To evaluate the possible involvement of infection, allergy and other immunological mechanisms in the pathophysiology of OME in children. Material and methods - Children selected to undergo myringotomy, were submitted to a questionnaire on middle ear disease history. Fluid effusion and serum samples weretested forthe measurement of immunoglobulins, specific IgE for food and common inhalant allergens, Tumor Necrosis Factor-.alpha. (TNF.alpha.), Interleukin-8 (II-8) and Eosinophil Cationic Protein (ECP). The microbiological study of effusion samples was also performed. Results - We studied 55 children, 35 male, mean age 7,1.+-.2,9 y, 18 (32,7%) with a personal and/ or family history of atopy. The serum laboratory study was done in 54 children. The IgE level was high in 18 (33,3%) and 6 (11,1%) had low levels of one or more immunoglobulins isotypes. Twenty (31,4%) were sensitized to food and/or inhalant allergens. We also detected high levels of ECP in 27 (52,9%), TNF.alpha. in 18 (33,3%) and II-8 was normal in all. In the effusion samples pathogenic bacteria were isolated in 18 (32,7%) and the levels of ECP, TNF.alpha. and 11-8 were higher than in serum. A significant correlation between serum IgE, ECP and cytokines and the effusion samples wasn't found. Discussion - In this study the inflammatory and ***allergic*** factors seem to beimportant in the etiopathogeny of OME. The deficiency of some immunoglobulin isotypes points to the need to exclude an immunodeficiency. The pattern of cytokines in the middle ear effusion reflects an intense local inflammatory reaction and the high levels of ECP suggests the participation of eosinophils.

L13 ANSWER 9 OF 20 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN
ACCESSION NUMBER: 2000-402970 [35] WPIDS
DOC. NO. CPI: C2000-122286
TITLE: Concentrated drink, for suppressing e.g. arthritis and ***allergic*** ***inflamation*** , consists of yeast fungus and lactic acid bacteria.
DERWENT CLASS: B04 D13 D16
PATENT ASSIGNEE(S): (YANG-N) YANG KK; (YANG-N) YANGU KK
COUNTRY COUNT: 2
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
JP 2000125810	A	20000509	(200035)*		9
JP 3276929	B2	20020422	(200234)		9
TW 555533	A	20031001	(200423)		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
JP 2000125810	A	JP 1998-320078	19981022
JP 3276929	B2	JP 1998-320078	19981022
TW 555533	A	TW 2000-107388	20000419

FILING DETAILS:

PATENT NO	KIND	PATENT NO
JP 3276929	B2 Previous Publ.	JP 2000125810

PRIORITY APPLN. INFO: JP 1998-320078 19981022
AN 2000-402970 [35] WPIDS
AB JP2000125810 A UPAB: 20030630
NOVELTY - Concentrated drink (I) consisting of yeast fungus and lactic acid bacteria, is new.

ACTIVITY - Anti-inflammatory; anti-arthritic; immunosuppressive. The concentrated drink was tested for anti-arthritic activity using rats induced with swelling using perfect Freund adjuvant. 1 hour after vaccination a concentrated drink was administered orally to the rat once a day for 5 days. A rat was also administered with 0.5% C.M.C. and (30mg/kg) of hydrocortisone. Swelling in the foot of the rat was reduced detectively and transition of inflammation was also suppressed by the concentrated drink.

MECHANISM OF ACTION - None given.

USE - (I) is useful for suppressing both chronic and acute inflammation such as in arthritis and ***allergic*** reactions. Dwg.0/0

L13 ANSWER 10 OF 20 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on STN
ACCESSION NUMBER: 2000:276647 BIOSIS
DOCUMENT NUMBER: PREV200000276647
TITLE: Apparatus for examining electrochemical effects of in vivo metal implants causing ***allergic*** symptoms and/or inflammation in a living organism.
AUTHOR(S): Vukan, Gyorgy [Inventor, Reprint author]; Vass, Zoltan [Inventor]; Krisko , Zoltan [Inventor]; Kiss, Laszlo [Inventor]; Sziraki, Laur [Inventor]; Varsanyi, Magda Lakatosne [Inventor]
CORPORATE SOURCE: Budapest, Hungary
ASSIGNEE: Dentimpex Kft., Budapest, Hungary
PATENT INFORMATION: US 5978692 November 02, 1999
SOURCE: Official Gazette of the United States Patent and Trademark Office Patents, (Nov. 2, 1999) Vol. 1228, No. 1. e-file. CODEN: OGUPE7. ISSN: 0098-1133.
DOCUMENT TYPE: Patent
LANGUAGE: English
ENTRY DATE: Entered STN: 6 Jul 2000
Last Updated on STN: 7 Jan 2002
AB An apparatus for examining the electrochemical effects of (in vivo) metal implants causing ***allergic*** symptoms and/or ***inflamation*** in living organism, the apparatus containing two probes and a signal processing circuit connected thereto. One of the probes is a reference electrode (1) provided with reference electrolyte (34), which is connected to the soma tissue near the implant while the other probe is a measuring electrode (2) provided with a metal contact tip (6) to be contacted with the implant. The reference electrode (1) and the measuring electrode (2) are connected through an amplifier (20, 27) to one of the inputs of a comparing unit (22, 28). The other input of the comparing unit (22, 28) is connected the output of a memory (24, 30) containing data concerning the metal to be examined, and one of the outputs of the comparing unit (22, 28) is connected the display for the measured data (11).

L13 ANSWER 11 OF 20 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN
ACCESSION NUMBER: 1998-348267 [30] WPIDS
DOC. NO. NON-CPI: N1998-271821
DOC. NO. CPI: C1998-107646
TITLE: Modulation of ***CD81*** -mediated signal transduction

- used for the treatment of e.g. ***allergic*** conditions, anaphylactic reactions, autoimmune disorders or bacterial or parasite infections.

DERWENT CLASS: B04 D16 S03
INVENTOR(S): FLEMING, T; KINET, J
PATENT ASSIGNEE(S): (BETH-N) BETH ISRAEL DEACONESS MEDICAL CENT; (FLEM-I) FLEMING T; (KINE-I) KINET J
COUNTRY COUNT: 22
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 9825647	A1	19980618	(199830)*	EN	62
RW: AT BE CH DE DK ES FI FR GB GR IE IT LU MC NL PT SE					
W: AU CA JP					
AU 9855220	A	19980703	(199847)		
EP 948354	A1	19991013	(199947)	EN	
R: AT BE CH DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE					
US 2002004210	A1	20020110	(200208)		
US 6423501	B2	20020723	(200254)		
US 2002182726	A1	20021205	(200301)		

APPLICATION DETAILS:

PATENT NO	KIND		APPLICATION	DATE
WO 9825647	A1		WO 1997-US22743	19971209
AU 9855220	A		AU 1998-55220	19971209
EP 948354	A1		EP 1997-951630	19971209
			WO 1997-US22743	19971209
US 2002004210	A1	Provisional	US 1996-32963P	19961213
			US 1997-954279	19971020
US 6423501	B2	Provisional	US 1996-32963P	19961213
			US 1997-954279	19971020
US 2002182726	A1	Provisional	US 1996-32963P	19961213
		Cont of	US 1997-954279	19971020
			US 2001-4562	20011205

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 9855220	A	Based on	WO 9825647
EP 948354	A1	Based on	WO 9825647

PRIORITY APPLN. INFO: US 1997-954279 19971020; US 1996-32963P 19961213; US 2001-4562 20011205

AN 1998-348267 [30] WPIDS
AB WO 9825647 A UPAB: 19991122

A calcium independent (CI) method of inhibiting cell surface receptor (CSR)-mediated signalling and/or ***degranulation*** (in a mammal), comprises contacting a cell (of the mammal) with an agent which induces ***CD81*** -mediated signal transduction (ST).

Inhibitors of ***CD81*** -mediated ST can be used conversely to enhance CSR-mediated signalling and/or ***degranulation*** .

USE - The methods can be used for the treatment of ***allergic*** conditions, e.g. asthma, hay fever or atopic eczema, anaphylactic reactions and related diseases. They can be used to treat ***allergic*** or ***inflammatory*** responses associated with disorders such as autoimmune diabetes mellitus, rheumatoid arthritis, ankylosing spondylitis, sarcoidosis, Sjogren's syndrome, multiple sclerosis, ***inflammatory*** bowel disease (i.e. Crohn's disease and ulcerative colitis), dermatomyositis, scleroderma, polymyositis, systemic lupus erythematosus, biliary cirrhosis, autoimmune thyroiditis, and autoimmune hepatitis, as well as many dermatological disorders, including psoriasis, contact sensitivity and atopic dermatitis. Enhancement of the cell surface receptors which induce mast cell ***degranulation*** is useful in inducing an ***inflammatory*** response, e.g. in response to bacterial or parasite infection. They can also be used to study receptor-mediated signalling in cells and to improve the therapeutic

capability to modulate the function of such cells.
Dwg.0/12

L13 ANSWER 12 OF 20 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 97368216 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9221759
TITLE: T helper 2 (Th2) T cells induce acute pancreatitis and diabetes in immune-compromised nonobese diabetic (NOD) mice.
AUTHOR: Pakala S V; Kurrer M O; Katz J D
CORPORATE SOURCE: Department of Pathology and Center for Immunology, Washington University School of Medicine, St. Louis, Missouri 63110, USA.
CONTRACT NUMBER: 1 P01 AI/DK 39676 (NIAID)
SOURCE: Journal of experimental medicine, (1997 Jul 21) 186 (2) 299-306.
Journal code: 2985109R. ISSN: 0022-1007.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; AIDS
ENTRY MONTH: 199708
ENTRY DATE: Entered STN: 19970825
Last Updated on STN: 19970825
Entered Medline: 19970814
AB Autoimmune diabetes is caused by the CD4(+), T helper 1 (Th1) cell-mediated apoptosis of insulin-producing beta cells. We have previously shown that Th2 T cells bearing the same T cell receptor (TCR) as the diabetogenic Th1 T cells invade islets in neonatal nonobese diabetic (NOD) mice but fail to cause disease. Moreover, when mixed in excess and cotransferred with Th1 T cells, Th2 T cells could not protect NOD neonates from Th1-mediated diabetes. We have now found, to our great surprise, the same Th2 T cells that produced a harmless insulitis in neonatal NOD mice produced intense and generalized pancreatitis and insulitis associated with islet cell necrosis, abscess formation, and subsequent diabetes when transferred into immunocompromised NOD.scid mice. These lesions resembled ***allergic*** ***inflamation*** and contained a large eosinophilic infiltrate. Moreover, the Th2-mediated destruction of islet cells was mediated by local interleukin-10 (IL-10) production but not by IL-4. These findings indicate that under certain conditions Th2 T cells may not produce a benign or protective insulitis but rather acute pathology and disease. Additionally, these results lead us to question the feasibility of Th2-based therapy in type I diabetes, especially in immunosuppressed recipients of islet cell transplants.

L13 ANSWER 13 OF 20 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1994:296052 CAPLUS
DOCUMENT NUMBER: 120:296052
TITLE: eosinophil differentiation and cytokine networks in ***allergic*** inflammation
AUTHOR(S): Denburg, Judah A.; Dolovich, Jerry; Marshall, Jean; Pin, Isabelle; Gibson, Peter; Ohno, Isao; Finotto, Susetta; Hargreave, Fred; Jordana, Manel
CORPORATE SOURCE: McMaster Univ., Hamilton, ON, Can.
SOURCE: Clinical Allergy and Immunology (1994), 2(Eosinophils in Allergy and Inflammation), 211-23
CODEN: CALMEH; ISSN: 1075-7910
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review and discussion, with 47 refs., on how eosinophil, mast cell, and basophil differentiation relates to the process of cell recruitment in ***allergic*** ***inflamation*** with emphasis on the role of corticosteroids and cytokines.

L13 ANSWER 14 OF 20 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on STN
ACCESSION NUMBER: 1991:245470 BIOSIS
DOCUMENT NUMBER: PREV199191126025; BA91:126025
TITLE: DIFFERENCES IN MIGRATION INHIBITORY FACTOR PRODUCTION BY C57BL-6 AND BALB-C MICE IN ***ALLERGIC*** AND IRRITANT CONTACT DERMATITIS.

AUTHOR(S): MALORNY U [Reprint author]; GOEBELER M; GUTWALD J; ROTH J; SORG C
CORPORATE SOURCE: INST EXP DERMATOL, UNIV MUENSTER, VON-ESMARCH-STRASSE 56, D-W-4400 MUENSTER, FRG
SOURCE: International Archives of Allergy and Applied Immunology, (1990) Vol. 92, No. 4, pp. 356-360.
CODEN: IAAAAM. ISSN: 0020-5915.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH
ENTRY DATE: Entered STN: 25 May 1991
Last Updated on STN: 16 Jul 1991

AB Two strains, of mice, BALB/c and C571/6, which are known to differ in their inflammatory responsiveness to allergens, were analyzed regarding their expression of macrophage migration inhibitory factor (MIF). ***Allergic*** contact dermatitis to 2,4-dinitro-1-fluorobenzene and irritant contact dermatitis to croton oil were studied immunohistologically at designated time intervals after elicitation. BALB/c mice presented a significantly more intense ear swelling response than C57Bl/6 mice and showed a strong endothelial MIF expression in the early phase of inflammatoin in both ***allergic*** and irritant contact dermatitis. Endothelial MIF expression was much weaker in C57B1/6 mice. Furthermore, the infiltration rate of inflammatory cells (MIF+ and BM8+ macrophages, Lyt2+ and L3T4+ T cells) was significantly higher in BALB/c than in C57B1/6 mice. We conclude that genetically determined differences of susceptibility to allergens and irritants in BALB/c and C57B1/6 mice are reflected by the intensity of MIF expression in the microvascular endothelium and immigrating inflammatory cells. MIF seems to appear as first molecular equivalent of developing ***inflamation*** and probably determines the degree of cellular infiltration.

L13 ANSWER 15 OF 20 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1981:624817 CAPLUS
DOCUMENT NUMBER: 95:224817
TITLE: Study of workers' skin from several departments in an oil industry in the city of Plock
AUTHOR(S): Ruszczak, Zdzislaw; Bienias, Ludomir; Proszynska-Kuczynska, Wieslawa
CORPORATE SOURCE: Klin. Dermatol., Wojsk. Akad.Med., Lodz, Pol.
SOURCE: Przeglad Lekarski (1981), 38(8), 637-9
CODEN: PRLKAV; ISSN: 0033-2240
DOCUMENT TYPE: Journal
LANGUAGE: Polish

AB A clin. study of 275 refinery and petrochem. industry workers revealed that 176 persons had various skin disorders. The most common complaint was the ***inflamation*** of the skin of feet. Also frequently obsd. were lichen pilaris, skin hyperkeratosis of feet and hands, melanodermia, and acne. The exposure to PhOH [108-95-2] produced addl. ***allergic*** (and ***allergic*** -toxic) dermatitis, while the exposure to AcPh [98-86-2] (in dewaxing) produced acne-like skin lesions.

L13 ANSWER 16 OF 20 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1979:483402 CAPLUS
DOCUMENT NUMBER: 91:83402
TITLE: Inhibition of ***allergic*** footpad inflammation by antirheumatic drug D-penicillamine
AUTHOR(S): Tsurufuji, Susumu; Ohuchi, Kazuo; Ishiguro, Masamichi; Miura, Mariko
CORPORATE SOURCE: Fac. Pharm. Sci., Tohoku Univ., Sendai, 980, Japan
SOURCE: Journal of Pharmacobio-Dynamics (1979), 2(3), 187-9
CODEN: JOPHDQ; ISSN: 0386-846X
DOCUMENT TYPE: Journal
LANGUAGE: English

AB An ***allergic*** footpad ***inflamation*** of mice was induced by using azobenzenearsonate-acetyl bovine serum albumin conjugate as an antigen. Sensitization was done with the aid of Freund's complete adjuvant and the ***allergic*** reaction was elicited by using an emulsion consisting of Freund's incomplete adjuvant and 0.9% NaCl soln. as a carrier of the challenging antigen. ***Allergic*** footpad swelling reached a max. 24 h after the challenge dose. D-Penicillamine [52-67-5] exerted a strong inhibitory effect on the ***allergic*** process, if

animals were treated with this drug for 21 days at a dose of 600 mg/kg/day.

L13 ANSWER 17 OF 20 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on STN
ACCESSION NUMBER: 1977:171728 BIOSIS
DOCUMENT NUMBER: PREV197763066592; BA63:66592
TITLE: IMMUNE AND BIOCHEMICAL MECHANISMS IN THE ***ALLERGIC*** DISEASE OF THE UPPER RESPIRATORY TRACT ROLE OF ANTIBODIES TARGET CELLS MEDIATORS AND EOSINOPHILS.
AUTHOR(S): HUBSCHER T T
SOURCE: Annals of Allergy, (1977) Vol. 38, No. 2, pp. 83-90. CODEN: ANAEA3. ISSN: 0003-4738.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: Unavailable
AB The pathways leading to the development of the ***allergic*** state and subsequently to the characteristic inflammatory response are complex and result from an interplay between immunologic and biochemical events. Several intrinsic factors, i.e., handling of antigens at mucosal level, transient immunodeficiency states (especially in the secretory IgA [immunoglobulin A] system), impairment in the IgE regulatory mechanism, modulation of cyclic nucleotides leading to mediator release and a feedback inhibition control provided by histamine and eosinophil derived products greatly dictate the outcome of events associated with ***allergic*** ***inflamation*** .

L13 ANSWER 18 OF 20 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on STN
ACCESSION NUMBER: 1976:213294 BIOSIS
DOCUMENT NUMBER: PREV197662043294; BA62:43294
TITLE: THE EPIDIDYMIS ASSOCIATED WITH GRANULOMATOUS ORCHITIS.
AUTHOR(S): KRUEGER R
SOURCE: Zentralblatt fuer Allgemeine Pathologie und Pathologische Anatomie, (1973) Vol. 117, No. 5-6, pp. 543-550. CODEN: ZAPPAN. ISSN: 0044-4030.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: Unavailable
AB Changes observed in 27 cases of granulomatous orchitis over a 4 yr period were described. Foci consisting of plasma cells and lymphocytes with productive fibrosis as in chronic ***inflamation*** of the epididymis were found in all cases. A granulomatous reaction, composed of macrophages, lymphocytes, plasma cells and polymorphonuclear leukocytes, in most cases arranged in the vicinity of ruptured ductuli efferents was noted in some inflammation cases. The inflammatory cells were found in a granular exudate, stained by the PAS (periodic acid-Schiff) method. Fibrosis was scarcely present. In 7 patients sperm were found. Interstitial granuloma consisting of macrophages, lymphocytes, plasma cells, multinuclear giant cells, polynuclear leukocytes, collagen and reticulin fibers comprised another type of inflammatory reaction. In 3 cases spermatic granulomas were present. Concentrically arranged fibrosis (pseudogranuloma), which results from destroyed ductuli and healed granulomas, were observed in cases dominated by small cells and fibrosis. Spermatozoa were seen in the interstitial tissue of the epididymis but could not be found in the tubuli or the interstitial tissue of the testicles. In granulomatous orchitis, extravasation of spermatozoa occurs only in the epididymis, never in the testicle. The extravasation of spermatozoa in the epididymis leads to direct contact with immune competent cells, lymphocytes and plasma cells. An antigen-antibody reaction is possible. Granulomatous orchitis could possibly be considered as an ***allergic*** reaction to sperm antibodies; the changes in the epididymis might be considered an inflammatory reaction to primary unspecific agents and secondary to the extravasation of spermatozoa.

L13 ANSWER 19 OF 20 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1963:477392 CAPLUS
DOCUMENT NUMBER: 59:77392
ORIGINAL REFERENCE NO.: 59:14460b-d
TITLE: Pharmacology of methdilazine. II. Some determinants and limits of action on vascular permeability and

inflammation in model systems
AUTHOR(S): Lish, Paul M.; McKinney, Gordon R.
CORPORATE SOURCE: Mead Johnson Res. Center, Evansville, IN
SOURCE: Journal of Laboratory and Clinical Medicine (1963), 61(6), 1015-28
CODEN: JLCMAK; ISSN: 0022-2143
DOCUMENT TYPE: Journal
LANGUAGE: Unavailable
AB Exptl. inflammatory and ***allergic*** reactions, as models of diseases of allergy and local inflammation, were used to study the pharmacology of methdilazine and certain other antiinflammatory, antiserotonin, and antihistamine drugs. Methdilazine opposed excessive permeability of capillaries, by its antihistamine, antiserotonin, and antibradykinin actions, plus an anti inflammatory action not related to any of these. Antiserotonin, antibradykinin, or antihistamine actions occurring separately or combined in a drug may be estd. through use of selected models of inflammation. The more conventional smooth muscle tests may similarly reveal those 3 activities, but quant. discrepancies may be apparent. It is suggested that capillary permeability tests are more indicative of possible drug utility. These expts. support the concept that mere antihistamine potency of a drug is not an adequate criterion for prediction of the drug's ultimate clin. value. Possession of a combination of such properties as antihistaminic, antiserotonin, antiphlogistic, and antibradykinin is indicated when the capillary endothelial cell is the target site of the agonist antagonist interaction. Certain drugs possess in various models of inflammation different degrees of specificity against the responsible mediators. At one extreme chlorpheniramine and LSD specifically inhibit edemas produced by histamine and serotonin, resp. These specific inhibitions contrast sharply with similarities of the inflammations induced by 2 tissue amines. At the opposite extreme aspirin generally inhibits edemas produced by the antigen antibody reaction and formalin, ultraviolet light induced erythema, and bradykinin induced wheals. Methdilazine falls in the middle of such a spectrum, showing good potency against histamine, serotonin, and bradykinin while still possessing the ability to antagonize such nonspecific insults as formalin and antigen antibody edemas and ultraviolet erythema.

L13 ANSWER 20 OF 20 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1962:10273 CAPLUS
DOCUMENT NUMBER: 56:10273
ORIGINAL REFERENCE NO.: 56:1930c-e
TITLE: Experimental study on the influence of various steroid substances upon ***allergic*** changes in the spleen
AUTHOR(S): Youn, Hal Byung
CORPORATE SOURCE: Ewha Woman's Univ., Seoul
SOURCE: Ch'oesin Uihak (1961), 4(No. 9), 45
CODEN: CHOUAX; ISSN: 0529-3804
DOCUMENT TYPE: Journal
LANGUAGE: Unavailable
AB Histol. study of the effects of cortisone, adrenocorticotropin (ACTH), estrogen and androgen in small and large doses on the extent of exptl. ***allergic*** inflammation induced in adult rabbit spleen showed that ACTH and especially cortisone exerted inhibitory effects on the ***allergic*** response. Small doses of cortisone were more effective than large while the reverse was true with ACTH. Estrogen was more effective than androgen in enhancing the ***allergic*** response. Large doses of both were more effective than small.

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L14 ANSWER 1 OF 18 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN DUPLICATE 1
ACCESSION NUMBER: 2004-064208 [07] WPIDS
DOC. NO. CPI: C2004-026367
TITLE: Inhibiting entry of malarial sporozoites into hepatocytes, useful for prevention of malaria, using an inhibitor of interaction between ***CD81*** and Plasmodium-associated ligand.
DERWENT CLASS: B04 D16

INVENTOR(S): BOUCHEIX, C; FRANETICH, J F; MAZIER, D; RUBINSTEIN, E; SILVIE, O
PATENT ASSIGNEE(S): (UYPA-N) UNIV CURIE PARIS VI P & M
COUNTRY COUNT: 1
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
FR 2840220	A1	20031205	(200407)*		20

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
FR 2840220	A1	FR 2002-6527	20020528

PRIORITY APPLN. INFO: FR 2002-6527 20020528
AN 2004-064208 [07] WPIDS
AB FR 2840220 A UPAB: 20040128
NOVELTY - Use of an inhibitor (I) of the interaction of ***CD81*** on hepatocytes with a Plasmodium-associated ligand (II) to prepare a composition for preventing infection by Plasmodium in humans, is new. (I) prevents penetration, as the result of formation of a functional parasitophore vacuole, of sporozoites into human hepatic cells.
DETAILED DESCRIPTION - An INDEPENDENT CLAIM is also included for an ex vivo or in vitro method for inhibiting penetration of Plasmodium sporozoites by inhibiting interaction between ***CD81*** and (II).
ACTIVITY - Protozoacide.
MECHANISM OF ACTION - Vaccine; inhibiting interaction between ***CD81*** on hepatocytes and ligands on the parasite.
CD81 is required for internalization by parasitophore vacuole formation and only sporozoites in the vacuoles can develop into schizonts and then into merozoites. Primary cultures of human hepatocytes were incubated with P. falciparum strain NF54, in presence of the anti-human ***CD81*** ***antibody*** 1D6. 3-4 days after infection, schizonts were detected with a labeled anti-heat-shock protein 70 antiserum. Treatment with 1D6 at 10 micro g/ml reduced the number of schizonts by 70-75 %.
USE - (I) is used to inhibit entry of Plasmodium falciparum, particularly, or P. vivax, P. malariae and P. ovale into liver cells. An agent (III) that induces formation of (I) in the treated host is administered as a prophylactic vaccine against malaria.
Dwg.0/0

L14 ANSWER 2 OF 18 MEDLINE on STN DUPLICATE 2
ACCESSION NUMBER: 2003270120 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12796480
TITLE: Tetraspanins CD9 and ***CD81*** function to prevent the fusion of mononuclear phagocytes.
AUTHOR: Takeda Yoshito; Tachibana Isao; Miyado Kenji; Kobayashi Masatoshi; Miyazaki Toru; Funakoshi Toshiki; Kimura Hiromi; Yamane Hiroyuki; Saito Yoshiyuki; Goto Hiroyuki; Yoneda Tsutomu; Yoshida Mitsuhiro; Kumagai Toru; Osaki Tadashi; Hayashi Seiji; Kawase Ichiro; Mekada Eisuke
CORPORATE SOURCE: Department of Molecular Medicine, Osaka University Graduate School of Medicine, Japan.
SOURCE: Journal of cell biology, (2003 Jun 9) 161 (5) 945-56. Journal code: 0375356. ISSN: 0021-9525.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200307
ENTRY DATE: Entered STN: 20030611
Last Updated on STN: 20030717
Entered Medline: 20030716
AB Tetraspanins CD9 and ***CD81*** facilitate the fusion between gametes, myoblasts, or virus-infected cells. Here, we investigated the role of these tetraspanins in the fusion of mononuclear phagocytes. Expression of CD9 and ***CD81*** and their complex formation with integrins were

up-regulated when blood monocytes were cultured under normal conditions. Under fusogenic conditions in the presence of Con A, CD9 and ***CD81*** up-regulation was inhibited, and their complex formation with integrins was down-regulated. Anti-CD9 and - ***CD81*** ***antibodies*** , which were previously shown to inhibit the fusion of gametes, myoblasts, and virus-infected cells, unexpectedly promoted the fusion of monocytes and alveolar macrophages. However, these effects were not due to altered cell adhesion, aggregation, or cytokine production. When stimulated in vitro or in vivo, alveolar macrophages and bone marrow cells of CD9- and ***CD81*** -null mice formed larger numbers of multinucleated cells than those of wild-type mice. Finally, CD9/ ***CD81*** double-null mice spontaneously developed multinucleated giant cells in the lung and showed enhanced osteoclastogenesis in the bone. These results suggest that CD9 and ***CD81*** coordinately prevent the fusion of mononuclear phagocytes.

L14 ANSWER 3 OF 18 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on STN
ACCESSION NUMBER: 2003:236059 BIOSIS
DOCUMENT NUMBER: PREV200300236059
TITLE: Binding of HCV E2 to ***CD81*** enhances surface expression of HLA-E.
AUTHOR(S): Nattermann, J. [Reprint Author]; Hofmeister, V.; Nischalke, H.-D. [Reprint Author]; Weiss, E.; Houghton, M.; Sauerbruch, T. [Reprint Author]; Spengler, U. [Reprint Author]
CORPORATE SOURCE: Department of Medicine I, University of Bonn, Bonn, Germany
SOURCE: Journal of Hepatology, (April 2003) Vol. 38, No. Supplement 2, pp. 116. print.
Meeting Info.: 38th Annual Meeting of the European Association for the Study of the Liver. Istanbul, Turkey. March 29-April 01, 2003. European Association for the Study of the Liver.
ISSN: 0168-8278 (ISSN print).
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 14 May 2003
Last Updated on STN: 14 May 2003

L14 ANSWER 4 OF 18 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on STN
ACCESSION NUMBER: 2003:236060 BIOSIS
DOCUMENT NUMBER: PREV200300236060
TITLE: Down-regulation of CCR5 expression by hepatitis C virus.
AUTHOR(S): Nattermann, J. [Reprint Author]; Nischalke, H.-D. [Reprint Author]; Houghton, M.; Sauerbruch, T. [Reprint Author]; Spengler, U. [Reprint Author]
CORPORATE SOURCE: Department of Medicine I, University of Bonn, Bonn, Germany
SOURCE: Journal of Hepatology, (April 2003) Vol. 38, No. Supplement 2, pp. 116. print.
Meeting Info.: 38th Annual Meeting of the European Association for the Study of the Liver. Istanbul, Turkey. March 29-April 01, 2003. European Association for the Study of the Liver.
ISSN: 0168-8278 (ISSN print).
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 14 May 2003
Last Updated on STN: 14 May 2003

L14 ANSWER 5 OF 18 MEDLINE on STN
ACCESSION NUMBER: 2003506275 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12597781
TITLE: Human ***CD81*** directly enhances Th1 and Th2 cell activation, but preferentially induces proliferation of Th2 cells upon long-term stimulation.
AUTHOR: Maecker Holden T
CORPORATE SOURCE: BD Biosciences, Immunocytometry Systems, 2350 Qume Drive, San Jose, CA 95131, USA.. holden_maecker@bd.com

SOURCE: BMC immunology [electronic resource], (2003 Feb 19) 4 (1) 1.
Journal code: 100966980. ISSN: 1471-2172.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200401
ENTRY DATE: Entered STN: 20031030
Last Updated on STN: 20040131
Entered Medline: 20040130

AB BACKGROUND: ***CD81*** , a cell-surface protein of the tetraspanin superfamily, has been shown to costimulate T cell activation in murine T cells, and is involved in development of Th2 immune responses in mice. RESULTS: Here it is shown that stimulation of ***CD81*** on human T cells can enhance T cell activation by antigen or superantigen, causing an increase in the early activation marker CD69, and increasing the number of cytokine-producing and proliferating T cells. Interestingly, ***CD81*** costimulates cytokine production by T cells producing both Th1 and Th2 cytokines. Although human ***CD81*** is highly expressed on non-T as well as T cells, ***CD81*** costimulation appears to act directly on T cells. Pre-incubation of purified T cells with ***anti*** - ***CD81*** ***antibody*** is sufficient to increase T cell activation, while pre-incubation of non-T cells is not. However, long-term polyclonal stimulation of T cells by anti-CD3 antibody, in the presence of ***CD81*** costimulation, biases T cells towards the production of IL-4 and not IFNgamma. This is accomplished by a preferential proliferation of IL-4-producing cells. CONCLUSION: Thus, signalling through ***CD81*** on T cells costimulates both Th1 and Th2 cells, but increases the number of Th2 cells during long-term activation.

L14 ANSWER 6 OF 18 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on STN DUPLICATE 3

ACCESSION NUMBER: 2003:184675 BIOSIS
DOCUMENT NUMBER: PREV200300184675
TITLE: Human ***CD81*** directly enhances Th1 and Th2 cell activation, but preferentially induces proliferation of Th2 cells upon long-term stimulation.
AUTHOR(S): Maecker, Holden T. [Reprint Author]
CORPORATE SOURCE: Immunocytometry Systems, BD Biosciences, 2350 Qume Drive, San Jose, CA, 95131, USA
holden_maecker@bd.com
SOURCE: BMC Immunology, (February 19 2003) Vol. 4, No. 1 Cited March 14, 2003. http://www.biomedcentral.com/1471-2172. online.
ISSN: 1471-2172 (ISSN online).
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 9 Apr 2003
Last Updated on STN: 9 Apr 2003

AB Background: ***CD81*** , a cell-surface protein of the tetraspanin superfamily, has been shown to costimulate T cell activation in murine T cells, and is involved in development of Th2 immune responses in mice. Results: Here it is shown that stimulation of ***CD81*** on human T cells can enhance T cell activation by antigen or superantigen, causing an increase in the early activation marker CD69, and increasing the number of cytokine-producing and proliferating T cells. Interestingly, ***CD81*** costimulates cytokine production by T cells producing both Th1 and Th2 cytokines. Although human ***CD81*** is highly expressed on non-T as well as T cells, ***CD81*** costimulation appears to act directly on T cells. Pre-incubation of purified T cells with ***anti*** - ***CD81*** ***antibody*** is sufficient to increase T cell activation, while pre-incubation of non-T cells is not. However, long-term polyclonal stimulation of T cells by anti-CD3 antibody, in the presence of ***CD81*** costimulation, biases T cells towards the production of IL-4 and not IFNgamma. This is accomplished by a preferential proliferation of IL-4-producing cells. Conclusion: Thus, signalling through ***CD81*** on T cells costimulates both Th1 and Th2 cells, but increases the number of Th2 cells during long-term activation.

L14 ANSWER 7 OF 18 MEDLINE on STN DUPLICATE 4

ACCESSION NUMBER: 2002078769 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11805134
TITLE: Primary hepatocytes of Tupaia belangeri as a potential model for hepatitis C virus infection.
AUTHOR: Zhao Xiping; Tang Zhen-Ya; Klumpp Bettina; Wolff-Vorbeck Guido; Barth Heidi; Levy Shoshana; von Weizsacker Fritz; Blum Hubert E; Baumert Thomas F
CORPORATE SOURCE: Department of Medicine II, University of Freiburg, Freiburg, Germany.
SOURCE: Journal of clinical investigation, (2002 Jan) 109 (2) 221-32.
Journal code: 7802877. ISSN: 0021-9738.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200202
ENTRY DATE: Entered STN: 20020128
Last Updated on STN: 20020220
Entered Medline: 20020219

AB Hepatitis C virus (HCV) is a major cause of chronic hepatitis worldwide, but the study of HCV infection has been hampered by the lack of an in vitro or in vivo small animal model. The tree shrew Tupaia belangeri is susceptible to infection with a variety of human viruses in vivo, including hepatitis viruses. We show that primary Tupaia hepatocytes can be infected with serum- or plasma-derived HCV from infected humans, as measured by de novo synthesis of HCV RNA, analysis of viral quasispecies evolution, and detection of viral proteins. Production of infectious virus could be demonstrated by passage to naive hepatocytes. To assess whether viral entry in Tupaia hepatocytes was dependent on the recently isolated HCV E2 binding protein ***CD81*** , we identified and characterized Tupaia ***CD81*** . Sequence analysis of cloned Tupaia cDNA revealed a high degree of homology between Tupaia and human ***CD81*** large extracellular loops (LEL). Cellular binding of E2 and HCV infection could not be inhibited by ***anti*** - ***CD81*** ***antibodies*** or soluble ***CD81*** -LEL, suggesting that viral entry can occur through receptors other than ***CD81*** . Thus, primary Tupaia hepatocytes provide a potential model for the study of HCV infection of hepatocytes.

L14 ANSWER 8 OF 18 MEDLINE on STN DUPLICATE 5
ACCESSION NUMBER: 2002453964 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12210409
TITLE: Cellular glycosaminoglycans and low density lipoprotein receptor are involved in hepatitis C virus adsorption.
AUTHOR: Germi Raphaele; Crance Jean-Marc; Garin Daniel; Guimet Josette; Lortat-Jacob Hugues; Ruigrok Rob W H; Zarski Jean-Pierre; Drouet Emmanuel
CORPORATE SOURCE: Laboratoire de Virologie Moleculaire et Structurale EA 2939, Universite Joseph Fourier, Faculte de Medecine-Pharmacie de Grenoble, La Tronche, France.
SOURCE: Journal of medical virology, (2002 Oct) 68 (2) 206-15.
Journal code: 7705876. ISSN: 0146-6615.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200210
ENTRY DATE: Entered STN: 20020906
Last Updated on STN: 20021031
Entered Medline: 20021030

AB The initial binding of Hepatitis C virus (HCV) to the cell membrane is a critical determinant of pathogenesis. Two putative HCV receptors have been identified, ***CD81*** and low-density lipoprotein receptor (LDLr). ***CD81*** interacts in vitro with the HCV E2 envelope glycoprotein, and LDLr interacts with HCV present in human plasma. In order to characterize these potential receptors for HCV, virus from plasma, able to replicate in cell culture, was inoculated on Vero cells or human hepatocarcinoma cells. HCV adsorption was assessed by quantitating cell-associated viral RNA by a real-time RT-PCR method. Anti-LDLr antibody, low and very low density lipoproteins inhibited significantly

HCV adsorption, confirming the role of LDLr as HCV receptor. Only one out of the two ***anti*** - ***CD81*** ***antibodies*** used in this study led to a partial inhibition of HCV binding. This study also highlights a role for glycosaminoglycans (GAGs) in HCV adsorption: treatment of virus with heparin led to 70% inhibition of attachment, as did desulfation of cellular GAGs. Treatment of Vero cells with heparin-lyase significantly inhibited virus attachment but by only 30%. These results demonstrate the complexity of the HCV binding step in which LDLr interacts strongly with HCV, whereas the interaction of HCV with GAGs and particularly with ***CD81*** seem to be more moderate.

L14 ANSWER 9 OF 18 MEDLINE on STN DUPLICATE 6
ACCESSION NUMBER: 2002055894 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11781363
TITLE: Inhibition of natural killer cells through engagement of ***CD81*** by the major hepatitis C virus envelope protein.
AUTHOR: Crotta Stefania; Stilla Annalisa; Wack Andreas; D'Andrea Annalisa; Nuti Sandra; D'Oro Ugo; Mosca Marta; Filliponi Franco; Brunetto R Maurizia; Bonino Ferruccio; Abrignani Sergio; Valiante Nicholas M
CORPORATE SOURCE: IRIS, Department of Immunology, Chiron S.p.A., 53100 Siena, Italy.
SOURCE: Journal of experimental medicine, (2002 Jan 7) 195 (1) 35-41.
Journal code: 2985109R. ISSN: 0022-1007.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200201
ENTRY DATE: Entered STN: 20020125
Last Updated on STN: 20020201
Entered Medline: 20020131

AB The immune response against hepatitis C virus (HCV) is rarely effective at clearing the virus, resulting in approximately 170 million chronic HCV infections worldwide. Here we report that ligation of an HCV receptor (***CD81***) inhibits natural killer (NK) cells. Cross-linking of ***CD81*** by the major envelope protein of HCV (HCV-E2) or ***anti*** - ***CD81*** ***antibodies*** blocks NK cell activation, cytokine production, cytotoxic granule release, and proliferation. This inhibitory effect was observed using both activated and resting NK cells. Conversely, on NK-like T cell clones, including those expressing NK cell inhibitory receptors, ***CD81*** ligation delivered a costimulatory signal. Engagement of ***CD81*** on NK cells blocks tyrosine phosphorylation through a mechanism which is distinct from the negative signaling pathways associated with NK cell inhibitory receptors for major histocompatibility complex class I. These results implicate HCV-E2-mediated inhibition of NK cells as an efficient HCV evasion strategy targeting the early antiviral activities of NK cells and allowing the virus to establish itself as a chronic infection.

L14 ANSWER 10 OF 18 BIOSIS on STN
ACCESSION NUMBER: 2003:283225 BIOSIS
DOCUMENT NUMBER: PREV200300283225
TITLE: ***Cd81*** REGULATES RETINAL PIGMENT EPITHELIA PROLIFERATION (CHANGES IN GENE EXPRESSION AND NULL MUTATION).
AUTHOR(S): Rogojina, A. T. [Reprint Author]; Song, B. K. [Reprint Author]; Geisert Jr, E. E. [Reprint Author]
CORPORATE SOURCE: Univ Tennessee, Memphis, TN, USA
SOURCE: Society for Neuroscience Abstract Viewer and Itinerary Planner, (2002) Vol. 2002, pp. Abstract No. 236.5. http://sfn.scholarone.com. cd-rom.
Meeting Info.: 32nd Annual Meeting of the Society for Neuroscience. Orlando, Florida, USA. November 02-07, 2002. Society for Neuroscience.
DOCUMENT TYPE: Conference; (Meeting)
Conference; (Meeting Poster)

Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 19 Jun 2003
Last Updated on STN: 19 Jun 2003

AB The present study focuses on the role of ***CD81*** (TAPA, the target of the antiproliferative antibody) in the regulation of the growth of retinal pigment epithelium (RPE). We examined the effects of the antibody on cultured cells and the consequences of a ***CD81*** null mutation on RPE number in the retina. RPE were cultured from Long-Evans rat pups. When RPE were cultured in the presence of ***anti*** - ***CD81*** ***antibody*** , the mitotic activity of the cells were depressed. This observation in tissue culture led us to examine the retina of mice with a ***CD81*** -null mutation. In the null mutation, there was a significant 10% increase in the number of RPE cells (student t test, $p < 0.012$). To further examine the mechanisms responsible for the control of proliferation by ***CD81*** , mRNA from cells treated with the ***anti*** - ***CD81*** ***antibody*** was isolated and compared to controls treated with control non-immune IgG. Each sample was a target for an Affymetrix Rat Chip (RG_U34A). Specific levels of mRNA were also confirmed using real time PCR. We confirmed the expression levels of selected proteins using immunoblot and immunohistochemical methods.)There was a significant change in 116 genes using the Mass 5 analysis from Affymetrix . The most intriguing changes were in genes regulating of cell cycle and second messenger systems. Previous studies demonstrated that ***CD81*** was expressed in retinal glia, the Muller cells that span the thickness of the retina, astrocytes found in the ganglion cell layer and RPE. Based on these results ***CD81*** appears to play an important role in regulating the number of RPE. We are currently analyzing the changes in gene expression that are associated with the antiproliferative effects of the antibodies.

L14 ANSWER 11 OF 18 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2001:816482 CAPLUS
DOCUMENT NUMBER: 135:356733
TITLE: Preparation of cell membrane vesicles and their potential uses
INVENTOR(S): Lamparski, Henry; Ruegg, Curtis; Le Pecq, Jean-Bernard; Hsu, Di-Hwei; Yao, Jenq-Yuan
PATENT ASSIGNEE(S): AP Cells, Inc., USA; Le Pecq, Jean-Bernard
SOURCE: PCT Int. Appl., 103 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001082958	A2	20011108	WO 2001-EP4173	20010411
WO 2001082958	A3	20020418		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
US 6812023	B1	20041102	US 2000-561205	20000427
US 2004241176	A1	20041202	US 2001-780748	20010209
CA 2407225	AA	20011108	CA 2001-2407225	20010411
EP 1278825	A2	20030129	EP 2001-943246	20010411
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
JP 2003531864	T2	20031028	JP 2001-579831	20010411
PRIORITY APPLN. INFO.:			US 2000-561205 A	20000427
			US 2001-780748 A	20010209
			WO 2001-EP4173 W	20010411

AB The present invention relates to methods of prepg. biol. material, and its various exptl., diagnostic, or therapeutic uses, including immunotherapy treatment or prophylaxis of tumors. More particularly, the present

invention relates to methods of prepg. membrane vesicles (in particular exosomes) released by various types of mammalian cells, comprising diafiltration and/or d. cushion centrifugation. The invention also provides novel methods for characterizing and analyzing exosome prepns., which can be used in quality control assay for the purpose of pharmaceutical product prodn. The invention is suitable to produce pharmaceutical grade prepns. of such membrane vesicles and to fully characterize said prepns., for use in humans. An example is presented wherein immature dendritic cells (DC), pulsed with a cytomegalovirus (CMV) peptide on the 5th day of cell culture, are used to produce peptide loaded exosomes (dexosomes). Dexosomes loaded with the CMV peptide specifically stimulated an anti-CMV T cell clone, and this required the presence of DC.

L14 ANSWER 12 OF 18 MEDLINE on STN DUPLICATE 7
ACCESSION NUMBER: 2001207053 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11240026
TITLE: ***CD81*** and microglial activation in vitro: proliferation, phagocytosis and nitric oxide production.
AUTHOR: Dijkstra S; Geisert E E Jr; Dijkstra C D; Bar P R; Joosten E A
CORPORATE SOURCE: Department of Experimental Neurology, UMC Utrecht, P.O. Box 85500, 3508 GA, Utrecht, The Netherlands.. s.dijkstra@neuro.azu.nl
SOURCE: Journal of neuroimmunology, (2001 Mar 1) 114 (1-2) 151-9. Journal code: 8109498. ISSN: 0165-5728.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200104
ENTRY DATE: Entered STN: 20010417
Last Updated on STN: 20010417
Entered Medline: 20010412
AB ***CD81*** (TAPA), a member of the tetraspanin family of proteins, is upregulated by astrocytes and microglia after traumatic injury to the rat central nervous system (CNS). To further understand the role of ***CD81*** in the microglial response to injury, we analysed the functional effects of a ***CD81*** ***antibody*** , AMP1, on cultured rat microglia. We found that AMP1 suppressed microglial proliferation in a dose-dependent manner. Furthermore, AMP1 stimulated myelin phagocytosis, probably by opsonizing the myelin. The phagocytosis of latex beads, as well as the production of nitric oxide, were not significantly influenced by AMP1. These data indicate that ***CD81*** is involved in an important subset of microglial effector functions after CNS injury.

L14 ANSWER 13 OF 18 MEDLINE on STN DUPLICATE 8
ACCESSION NUMBER: 2000472791 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10806098
TITLE: Transmembrane-4-superfamily proteins CD151 and ***CD81*** associate with alpha 3 beta 1 integrin, and selectively contribute to alpha 3 beta 1-dependent neurite outgrowth.
AUTHOR: Stipp C S; Hemler M E
CORPORATE SOURCE: Department of Cancer Immunology and AIDS, Dana-Farber Cancer Institute and Department of Pathology, Harvard Medical School, Boston, MA 02115, USA.
CONTRACT NUMBER: GM38903 (NIGMS)
NS10344 (NINDS)
SOURCE: Journal of cell science, (2000 Jun) 113 (Pt 11) 1871-82. Journal code: 0052457. ISSN: 0021-9533.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200010
ENTRY DATE: Entered STN: 20001012
Last Updated on STN: 20001012
Entered Medline: 20001005
AB Proteins in the transmembrane-4-superfamily (TM4SF) form many different complexes with proteins in the integrin family, but the functional utility of these complexes has not yet been demonstrated. Here we show that TM4SF

proteins CD151, ***CD81*** , and CD63 co-distribute with alpha3beta1 integrin on neurites and growth cones of human NT2N cells. Also, stable CD151-alpha3beta1 and ***CD81*** -alpha3beta1 complexes were recovered in NT2N detergent lysates. Total NT2N neurite outgrowth on laminin-5 (a ligand for alpha3beta1 integrin) was strongly inhibited by anti-CD151 and - ***CD81*** ***antibodies*** either together (approximately 85% inhibition) or alone (approximately 45% inhibition). Notably, these antibodies had no inhibitory effect on NT2N neurites formed on laminin-1 or fibronectin, when alpha3beta1integrin was not engaged. Neurite number, length, and rate of extension were all affected by anti-TM4SF antibodies. In summary: (1) these substrate-dependent inhibition results strongly suggest that CD151 and ***CD81*** associations with alpha3beta1 are functionally relevant, (2) TM4SF proteins CD151 and ***CD81*** make a strong positive contribution toward neurite number, length, and rate of outgrowth, and (3) NT2N cells, a well-established model of immature central nervous system neurons, can be a powerful system for studies of integrin function in neurite outgrowth and growth cone motility.

L14 ANSWER 14 OF 18 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on STN

ACCESSION NUMBER: 2001:76814 BIOSIS
DOCUMENT NUMBER: PREV200100076814
TITLE: Intraparenchymal infusion of anti-TAPA/ ***CD81*** ***antibodies*** leads to functional recovery after spinal cord injury.
AUTHOR(S): Hamers, F. P. [Reprint author]; Dijkstra, S.; Lankhorst, A. J.; Joosten, E. A.; Bar, P. R.; Gispen, W. H.; Geisert, E. E., Jr.
CORPORATE SOURCE: Rudolf Magnus Institute for Neurosciences, University Medical Center, Utrecht, Netherlands
SOURCE: Society for Neuroscience Abstracts, (2000) Vol. 26, No. 1-2, pp. Abstract No.-186.17. print.
Meeting Info.: 30th Annual Meeting of the Society of Neuroscience. New Orleans, LA, USA. November 04-09, 2000. Society for Neuroscience.
ISSN: 0190-5295.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 7 Feb 2001
Last Updated on STN: 12 Feb 2002

AB Modulation of the glial response to spinal cord injury may lead to enhanced functional recovery. The monoclonal antibody AMP1 was found to alter the stability of astrocyte-astrocyte contact in vitro and to inhibit proliferation of astrocytes and microglia. Furthermore, the AMP1 antigen (TAPA/ ***CD81***) is upregulated after traumatic spinal cord injury. Therefore we studied whether intralesional infusion of AMP1-mAb could enhance functional recovery after spinal cord contusion injury. Female Wistar rats were subjected to a moderate spinal cord contusion injury and implanted at the lesion site with a stainless steel cannula connected to an osmotic minipump. Two different doses of AMP1-mAb and one dose of pre-immune IgG were infused for 14 days. Neurological function was regularly assessed on several function tests for 8 weeks. The lower dose of AMP1 led to significantly better function on BBB (+-1.5 point) and Gridwalk tests as compared to the IgG control from 3 weeks onward. Hindpaw fine motor function, as assessed by BBB-subscores, was significantly better from 2 weeks onward. The higher dose of AMP1 did not differ from IgG control. These data suggest that AMP1 might be of value in the treatment of spinal cord injury, either by modulating the primary inflamatory process or by affecting the formation of the glial scar.

L14 ANSWER 15 OF 18 MEDLINE on STN DUPLICATE 9

ACCESSION NUMBER: 1999389749 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10459022
TITLE: Role of transmembrane 4 superfamily (TM4SF) proteins CD9 and ***CD81*** in muscle cell fusion and myotube maintenance.
AUTHOR: Tachibana I; Hemler M E
CORPORATE SOURCE: Dana-Farber Cancer Institute, and Harvard Medical School, Boston, Massachusetts 02115, USA.
CONTRACT NUMBER: GM38903 (NIGMS)

SOURCE: Journal of cell biology, (1999 Aug 23) 146 (4) 893-904.
Journal code: 0375356. ISSN: 0021-9525.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199909
ENTRY DATE: Entered STN: 19991005
Last Updated on STN: 19991005
Entered Medline: 19990923

AB The role of transmembrane 4 superfamily (TM4SF) proteins during muscle cell fusion has not been investigated previously. Here we show that the appearance of TM4SF protein, CD9, and the formation of CD9-beta1 integrin complexes were both regulated in coordination with murine C2C12 myoblast cell differentiation. Also, anti-CD9 and anti- ***CD81*** monoclonal antibodies substantially inhibited and delayed conversion of C2C12 cells to elongated myotubes, without affecting muscle-specific protein expression. Studies of the human myoblast-derived RD sarcoma cell line further demonstrated that TM4SF proteins have a role during muscle cell fusion. Ectopic expression of CD9 caused a four- to eightfold increase in RD cell syncytia formation, whereas anti-CD9 and ***anti*** - ***CD81*** ***antibodies*** markedly delayed RD syncytia formation. Finally, anti-CD9 and anti- ***CD81*** monoclonal antibodies triggered apoptotic degeneration of C2C12 cell myotubes after they were formed. In summary, TM4SF proteins such as CD9 and ***CD81*** appear to promote muscle cell fusion and support myotube maintenance.

L14 ANSWER 16 OF 18 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1998:402338 CAPLUS
DOCUMENT NUMBER: 129:66839
TITLE: Calcium-independent modulation by ***CD81*** of receptor signalling
INVENTOR(S): Fleming, Tony; Kinet, Jean-Pierre
PATENT ASSIGNEE(S): Beth Israel Deaconess Medical Center, USA
SOURCE: PCT Int. Appl., 63 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9825647	A1	19980618	WO 1997-US22743	19971209
W: AU, CA, JP				
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
US 2002004210	A1	20020110	US 1997-954279	19971020
US 6423501	B2	20020723		
AU 9855220	A1	19980703	AU 1998-55220	19971209
EP 948354	A1	19991013	EP 1997-951630	19971209
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
US 2002182726	A1	20021205	US 2001-4562	20011205

PRIORITY APPLN. INFO.: US 1996-32963P P 19961213
US 1997-954279 A 19971020
WO 1997-US22743 W 19971209

AB Calcium-independent ***CD81*** inhibition of IgE-mediated degranulation in mast cells, particularly through the Fc.gamma.RIII and Fc.epsilon.RI receptors, is described, as well as methods of inhibiting allergic processes. The method uses monoclonal ***anti*** - ***CD81*** ***antibody*** .

REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER 17 OF 18 MEDLINE on STN DUPLICATE 10
ACCESSION NUMBER: 97477414 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9334370
TITLE: Negative regulation of Fc epsilon RI-mediated degranulation by ***CD81*** .
AUTHOR: Fleming T J; Donnadieu E; Song C H; Laethem F V; Galli S J; Kinet J P

CORPORATE SOURCE: Department of Pathology, Beth Israel Deaconess Medical Center, Boston, Massachusetts 02215, USA.
CONTRACT NUMBER: AI/CA-23990 (NIAID)
CA/AI-72074 (NCI)
GM-53950 (NIGMS)
SOURCE: Journal of experimental medicine, (1997 Oct 20) 186 (8) 1307-14.
Journal code: 2985109R. ISSN: 0022-1007.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199711
ENTRY DATE: Entered STN: 19971224
Last Updated on STN: 19971224
Entered Medline: 19971121

AB Signaling through the high affinity receptor for immunoglobulin E (Fc epsilon RI) results in the coordinate activation of tyrosine kinases before calcium mobilization. Receptors capable of interfering with the signaling of antigen receptors, such as Fc epsilon RI, recruit tyrosine and inositol phosphatases that results in diminished calcium mobilization. Here, we show that antibodies recognizing ***CD81*** inhibit Fc epsilon RI-mediated mast cell degranulation but, surprisingly, without affecting aggregation-dependent tyrosine phosphorylation, calcium mobilization, or leukotriene synthesis. Furthermore, ***CD81*** ***antibodies*** also inhibit mast cell degranulation in vivo as measured by reduced passive cutaneous anaphylaxis responses. These results reveal an unsuspected calcium-independent pathway of antigen receptor regulation, which is accessible to engagement by membrane proteins and on which novel therapeutic approaches to allergic diseases could be based.

L14 ANSWER 18 OF 18 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1997:102960 CAPLUS
DOCUMENT NUMBER: 126:116790
TITLE: ***CD81*** (TAPA-1), a component of the CD19/CD21 signal transduction complex
AUTHOR(S): Bradbury, Laura; Tedder, Thomas F.
CORPORATE SOURCE: UK
SOURCE: Leucocyte Typing V: White Cell Differentiation Antigens, Proceedings of the International Workshop and Conference, 5th, Boston, Nov. 3-7, 1993 (1995), Meeting Date 1993, Volume 1, 690-691. Editor(s): Schlossman, Stuart F. Oxford University Press: Oxford, UK.
CODEN: 63WDAC
DOCUMENT TYPE: Conference
LANGUAGE: English

AB ***CD81*** is a member of the tetraspans family of cell surface mols. which exists on the cell surface as a part of a multimeric signaling complex, that, in the case of B-cell lines, may include CD19, CD21, Leu 13, MHC class II proteins, and other undefined proteins. A panel of anti-B-cell monoclonal antibodies were characterized. At least 2 non-overlapping epitopes were defined by the ***anti*** - ***CD81*** ***antibodies*** examd. here, and these epitopes were not equiv. in their ability to induce transmembrane signaling via the ***CD81*** protein. However, there were no differences in the ability of these monoclonal antibodies to co-ppt. the ***CD81*** complex from the surface of B-cell lines.

=> d ibib abs l15 1-2

L15 ANSWER 1 OF 2 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1998:402338 CAPLUS
DOCUMENT NUMBER: 129:66839
TITLE: Calcium-independent modulation by ***CD81*** of receptor signalling
INVENTOR(S): Fleming, Tony; Kinet, Jean-Pierre
PATENT ASSIGNEE(S): Beth Israel Deaconess Medical Center, USA
SOURCE: PCT Int. Appl., 63 pp.

CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9825647	A1	19980618	WO 1997-US22743	19971209
W: AU, CA, JP				
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
US 2002004210	A1	20020110	US 1997-954279	19971020
US 6423501	B2	20020723		
AU 9855220	A1	19980703	AU 1998-55220	19971209
EP 948354	A1	19991013	EP 1997-951630	19971209
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
US 2002182726	A1	20021205	US 2001-4562	20011205
PRIORITY APPLN. INFO.:			US 1996-32963P P	19961213
			US 1997-954279 A	19971020
			WO 1997-US22743 W	19971209

AB Calcium-independent ***CD81*** inhibition of IgE-mediated ***degranulation*** in mast cells, particularly through the Fc.gamma.RIII and Fc.epsilon.RI receptors, is described, as well as methods of inhibiting allergic processes. The method uses monoclonal ***anti*** - ***CD81*** ***antibody*** .

REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 2 OF 2 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 97477414 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9334370
TITLE: Negative regulation of Fc epsilon RI-mediated ***degranulation*** by ***CD81*** .
AUTHOR: Fleming T J; Donnadieu E; Song C H; Laethem F V; Galli S J; Kinet J P
CORPORATE SOURCE: Department of Pathology, Beth Israel Deaconess Medical Center, Boston, Massachusetts 02215, USA.
CONTRACT NUMBER: AI/CA-23990 (NIAID)
CA/AI-72074 (NCI)
GM-53950 (NIGMS)
SOURCE: Journal of experimental medicine, (1997 Oct 20) 186 (8) 1307-14.
Journal code: 2985109R. ISSN: 0022-1007.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199711
ENTRY DATE: Entered STN: 19971224
Last Updated on STN: 19971224
Entered Medline: 19971121

AB Signaling through the high affinity receptor for immunoglobulin E (Fc epsilon RI) results in the coordinate activation of tyrosine kinases before calcium mobilization. Receptors capable of interfering with the signaling of antigen receptors, such as Fc epsilon RI, recruit tyrosine and inositol phosphatases that results in diminished calcium mobilization. Here, we show that antibodies recognizing ***CD81*** inhibit Fc epsilon RI-mediated mast cell ***degranulation*** but, surprisingly, without affecting aggregation-dependent tyrosine phosphorylation, calcium mobilization, or leukotriene synthesis. Furthermore, ***CD81*** ***antibodies*** also inhibit mast cell ***degranulation*** in vivo as measured by reduced passive cutaneous anaphylaxis responses. These results reveal an unsuspected calcium-independent pathway of antigen receptor regulation, which is accessible to engagement by membrane proteins and on which novel therapeutic approaches to allergic diseases could be based.

=> d ibib abs l17 1-2

L17 ANSWER 1 OF 2 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1998:402338 CAPLUS
DOCUMENT NUMBER: 129:66839
TITLE: Calcium-independent modulation by ***CD81*** of receptor signalling
INVENTOR(S): Fleming, Tony; Kinet, Jean-Pierre
PATENT ASSIGNEE(S): Beth Israel Deaconess Medical Center, USA
SOURCE: PCT Int. Appl., 63 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9825647	A1	19980618	WO 1997-US22743	19971209
W: AU, CA, JP				
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
US 2002004210	A1	20020110	US 1997-954279	19971020
US 6423501	B2	20020723		
AU 9855220	A1	19980703	AU 1998-55220	19971209
EP 948354	A1	19991013	EP 1997-951630	19971209
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
US 2002182726	A1	20021205	US 2001-4562	20011205

PRIORITY APPLN. INFO.: US 1996-32963P P 19961213
US 1997-954279 A 19971020
WO 1997-US22743 W 19971209

AB Calcium-independent ***CD81*** inhibition of IgE-mediated degranulation in mast cells, particularly through the Fc.gamma.RIII and Fc.epsilon.RI receptors, is described, as well as methods of inhibiting allergic processes. The method uses monoclonal anti- ***CD81*** antibody.

REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L17 ANSWER 2 OF 2 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 97477414 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9334370
TITLE: Negative regulation of Fc epsilon RI-mediated degranulation by ***CD81*** .
AUTHOR: Fleming T J; Donnadieu E; Song C H; Laethem F V; Galli S J; Kinet J P
CORPORATE SOURCE: Department of Pathology, Beth Israel Deaconess Medical Center, Boston, Massachusetts 02215, USA.
CONTRACT NUMBER: AI/CA-23990 (NIAID)
CA/AI-72074 (NCI)
GM-53950 (NIGMS)
SOURCE: Journal of experimental medicine, (1997 Oct 20) 186 (8) 1307-14.
Journal code: 2985109R. ISSN: 0022-1007.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199711
ENTRY DATE: Entered STN: 19971224
Last Updated on STN: 19971224
Entered Medline: 19971121

AB Signaling through the high affinity receptor for immunoglobulin E (Fc epsilon RI) results in the coordinate activation of tyrosine kinases before calcium mobilization. Receptors capable of interfering with the signaling of antigen receptors, such as Fc epsilon RI, recruit tyrosine and inositol phosphatases that results in diminished calcium mobilization. Here, we show that antibodies recognizing ***CD81*** inhibit Fc epsilon RI-mediated mast cell degranulation but, surprisingly, without affecting aggregation-dependent tyrosine phosphorylation, calcium mobilization, or leukotriene synthesis. Furthermore, ***CD81*** antibodies also inhibit mast cell degranulation in vivo as measured by reduced ***passive*** ***cutaneous*** ***anaphylaxis***

responses. These results reveal an unsuspected calcium-independent pathway of antigen receptor regulation, which is accessible to engagement by membrane proteins and on which novel therapeutic approaches to allergic diseases could be based.

=> d ibib abs l16

L16 ANSWER 1 OF 1 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN DUPLICATE 1
ACCESSION NUMBER: 1998-348267 [30] WPIDS
DOC. NO. NON-CPI: N1998-271821
DOC. NO. CPI: C1998-107646
TITLE: Modulation of ***CD81*** -mediated signal transduction - used for the treatment of e.g. allergic conditions, anaphylactic reactions, autoimmune disorders or bacterial or parasite infections.
DERWENT CLASS: B04 D16 S03
INVENTOR(S): FLEMING, T; KINET, J
PATENT ASSIGNEE(S): (BETH-N) BETH ISRAEL DEACONESS MEDICAL CENT; (FLEM-I) FLEMING T; (KINE-I) KINET J
COUNTRY COUNT: 22
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 9825647	A1	19980618	(199830)*	EN	62
RW: AT BE CH DE DK ES FI FR GB GR IE IT LU MC NL PT SE					
W: AU CA JP					
AU 9855220	A	19980703	(199847)		
EP 948354	A1	19991013	(199947)	EN	
R: AT BE CH DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE					
US 2002004210	A1	20020110	(200208)		
US 6423501	B2	20020723	(200254)		
US 2002182726	A1	20021205	(200301)		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 9825647	A1	WO 1997-US22743	19971209
AU 9855220	A	AU 1998-55220	19971209
EP 948354	A1	EP 1997-951630	19971209
		WO 1997-US22743	19971209
US 2002004210	A1 Provisional	US 1996-32963P	19961213
		US 1997-954279	19971020
US 6423501	B2 Provisional	US 1996-32963P	19961213
		US 1997-954279	19971020
US 2002182726	A1 Provisional	US 1996-32963P	19961213
	Cont of	US 1997-954279	19971020
		US 2001-4562	20011205

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 9855220	A Based on	WO 9825647
EP 948354	A1 Based on	WO 9825647

PRIORITY APPLN. INFO: US 1997-954279 19971020; US 1996-32963P 19961213; US 2001-4562 20011205
AN 1998-348267 [30] WPIDS
AB WO 9825647 A UPAB: 19991122
A calcium independent (CI) method of inhibiting cell surface receptor (CSR)-mediated signalling and/or degranulation (in a mammal), comprises contacting a cell (of the mammal) with an agent which induces ***CD81*** -mediated signal transduction (ST).

Inhibitors of ***CD81*** -mediated ST can be used conversely to enhance CSR-mediated signalling and/or degranulation.

USE - The methods can be used for the treatment of allergic conditions, e.g. asthma, ***hay*** ***fever*** or ***atopic***

eczema , anaphylactic reactions and related diseases. They can be used to treat allergic or inflammatory responses associated with disorders such as autoimmune diabetes mellitus, rheumatoid arthritis, ankylosing spondylitis, sarcoidosis, Sjogren's syndrome, multiple sclerosis, inflammatory bowel disease (i.e. Crohn's disease and ulcerative colitis), dermatomyositis, scleroderma, polymyositis, systemic lupus erythematosus, biliary cirrhosis, autoimmune thyroiditis, and autoimmune hepatitis, as well as many dermatological disorders, including psoriasis, contact sensitivity and atopic dermatitis. Enhancement of the cell surface receptors which induce mast cell degranulation is useful in inducing an inflammatory response, e.g. in response to bacterial or parasite infection. They can also be used to study receptor-mediated signalling in cells and to improve the therapeutic capability to modulate the function of such cells.
Dwg.0/12

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